Original Article

Lumbar Spondylosis: Clinical Presentation And Treatment Approaches – A Systematic Review

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Abstract

Low back pain (LBP) affects approximately 60–85% of adults during some point in their lives. Fortunately, for the large majority of individuals, symptoms are mild and transient, with 90% subsiding within 6 weeks. Chronic low back pain, defined as pain symptoms persisting beyond 3 months, affects an estimated 15–45% of the population. For the minority with intractable symptoms, the impact on quality of life and economic implications are considerable. Despite the high prevalence of low back pain within the general population, the diagnostic approach and therapeutic options are diverse and often inconsistent, resulting in rising costs and variability in management throughout the country. In part, this is due to the difficulty establishing a clear etiology for most patients, with known nociceptive pain generators identified throughout the axial spine. Back pain has been termed as ‘‘an illness in search of a disease.’’ Indeed, once ‘‘red flag’’ diagnoses such as cancer and fracture have been ruled out, the differential sources of low back pain remain broad, including the extensive realm of degenerative changes within the axial spine for which radiological evaluation is nonspecific and causal relationships are tentative. We will elaborate on these degenerative processes and their clinical implications. We will further discuss diagnostic approaches and the efficacy of existing treatment options.

Keywords Osteophyte, Low back pain, Spondylosis, Intervertebral osteochondrosis, Degenerative disk disease

Introduction

At some point in their life, 60 to 85% of adults will experience low back pain (LBP) [1-3]. Fortunately, 90% of people experience moderate and short-lived symptoms, which go away within 6 weeks [4]. An estimated 15 to 45% of the population suffers from chronic low back pain, which is characterised as symptoms that continue for longer than three months [5, 6]. The impact on quality of life and the financial implications are significant for the minority of people with intractable symptoms [7]. Despite the significant incidence of low back pain in the general population, there are numerous, frequently inconclusive therapeutic alternatives and diagnostic approaches, which drive up prices and provide regional variations in management [8]. Since known nociceptive pain generators have been found everywhere along the axial spine [9], it might be challenging to determine a definite aetiology for the majority of patients. Back discomfort has been referred to as "a disease in search of an illness" [10]. After "red flag" diagnoses like cancer and fracture have been ruled out, the differential causes of low back pain are still wide-ranging and include the extensive field of degenerative changes within the axial spine, for which radiological evaluation is nonspecific and causal relationships are uncertain [11, 12-18]. We will elaborate on these

degenerative processes and their clinical implications. We will further discuss the diagnostic approaches and the efficacy of existing treatment options.

Epidemiology

In population studies, degenerative spine alterations are strikingly prevalent. 85.5% of participants in Symmons et al[19] .'s research of people between the ages of 45 and 64 showed osteophytes in the lumbar spine. In a study by O'Neill et al. [20] on osteophytosis in UK adults over 50, it was discovered that 84% of men and 74% of women had at least one spinal osteophyte, with incidence rates being greater in people who were more physically active, reported having back discomfort, or had higher BMI scores. Men appear to experience more profound degenerative alterations than women do, both in terms of the quantity and severity of osteophyte production, despite wide variation within the population [20].

It is impressive to see radiographic evidence of lumbar spine degeneration in asymptomatic people. 80% [21] of asymptomatic patients over 60 years old with MRI imaging had disc protrusions, and 20% [11] have degenerative spinal stenosis. In a research evaluating bone spurs and disc space narrowing among males with no pain, moderate discomfort, or severe lower back pain, radiographic evidence of spine degeneration was detected in all three groups with a similar frequency [22].

Furthermore, degenerative changes may appear in young individuals without decades of spine loading. Lawrence [23] found 10% of women aged 20–29 to demonstrate evidence of disk degeneration. Lumbar spondylosis, while affecting 80% of patients older than 40 years, nevertheless was found in 3% of individuals aged 20–29 years in one study [15]. The high incidence of degeneration among young and asymptomatic individuals highlights the challenge involved in establishing causality between imaging findings and pain symptoms in affected patients.

Pathogenesis

The frequent occurrence of intervertebral disc, vertebral body, and related joints experiencing degenerative changes at the same time raises the possibility of a progressive and dynamic mechanism, with interdependent changes developing as a result of disc space narrowing [17]. According to Kirkaldy Willis and Bernard [24], intervertebral discs go through a "degenerative cascade" (Fig. 1), which consists of three overlapping phases and may take place over many years. Phase I (Dysfunction Phase) outlines the early consequences of repeated microtrauma with the emergence of circumferential painful tears of the outer, innervated anulus and related endplate separation that may impair the nutritional supply and waste clearance of the disc. These rips may combine to form radial tears, which are more likely to protrude, and which affect the disk's ability to retain water, leading to desiccation and a decreased disc height. Vascular tissue and nerve endings may grow into fissures, boosting innervation and the disk's ability to transmit pain signals [25]. Phase II (Instability Phase) is characterised by the loss of mechanical integrity, progressive disc alterations, including internal disruption, more annular rips, and disc resorption, along with further facet degeneration that may lead to instability and subluxation. Phase III (Stabilization Phase) is characterised by persistent fibrosis and constriction of the disc space, osteophyte development, and transdiscal bridging [26].

In order to explain other degenerative alterations of the axial spine, Schneck offers a further mechanical progression that builds on this intervertebral disc degeneration cascade. He suggests numerous effects of disc space contraction. A narrowing of the superior-inferior dimension of the intervertebral canal occurs between adjacent pedicles. The ligamentum flavum can bulge and the possibility of spine instability is made possible by longitudinal ligament laxity caused by minimal redundancy. The superior articular process (SAP) can sublux due to increased spine movement, which results in a smaller anteroposterior dimension of the intervertebral and upper nerve root canals. Laxity may also result in altered weight-bearing mechanisms and pressure relationships on vertebral bones and joint spaces, which are thought to influence the development of osteophytes and facet hypertrophy on both inferior and superior articular processes with potential risks for projection into the intervertebral canal and central canal, respectively. The spinal canal, nerve root canal, and intervertebral canal may become anteriorly encroached upon as a result of oblique orientations of the articular processes, which may also result in retrospondylolisthesis [17, 27-29].

Clinical presentation

It is not unexpected that the axial spine's facet joints, intervertebral discs, sacroiliac joints, nerve root dura, and myofascial structures are nociceptive pain producers at the site of these degenerative alterations [9]. Through progressive ingrowth of osteophytes, hypertrophy of the inferior articular process [31], disc herniation, bulging of the ligamentum flavum [17], or spondylolisthesis, these degenerative anatomical changes may result in a clinical presentation of spinal stenosis, or narrowing within the spinal canal [30]. A number of pain symptoms, together referred to as neurogenic claudication, are the clinical outcome (NC). Lower back discomfort, leg pain, numbness, and motor weakness in the lower extremities are possible symptoms of NC, to varied degrees [30–35]. These symptoms tend to get worse with standing up and walking and get better while laying down.

Etiology/risk factors

The influence of age

The biggest risk factor for bone degradation, notably in the spine, has long been acknowledged by large investigations of osteoarthritis [36]. According to a thorough autopsy investigation conducted in 1926, spondylitis deformans evidence increased linearly between the ages of 39 and 70 years, from 0% to 72% [37]. According to Miller et alfollowing .'s autopsy investigation [38], which used macroscopic disc degeneration grades from 600 specimens, disc degeneration increased from 16% at age 20 to nearly 98% at age 70. This result is supported by additional research [20, 39]. Even still, the correlations are not flawless. In a retrospective analysis of radiographs of women, Kramer [40] discovered that growing older was substantially linked with osteophyte production but was not predictive of the level of disc space narrowing. Although few younger women have high average scores, she saw significant variation, stating that "some older women show no radiographic indication of OA, whereas others are badly impacted."

The impact of activity and occupation

Disk formation has historically been linked to specific actions. Body Mass Index (BMI), incident back trauma, daily spine loading (twisting, lifting, bending, and persistent nonneutral postures), and whole body vibration (like vehicle driving) are all factors that raise the risk and severity of spondylosis, according to retrospective research [20, 41]. Even though these correlations exist, a study tracking progressive radiographic changes in lumber DDD found no evidence of a significant relationship between physical activity level and DDD or osteophyte changes [42]. Instead, it found only that age, back pain, and associated hip OA are predictors of DDD and osteophyte changes. The function of heredity Osteophytes and disc degeneration are probably influenced by genetic factors. According to Spector and MacGregor [43], heritable variables account for 50% of the variation in osteoarthritis. Similar to this, twin studies examining the evolution of degenerative alterations in lumbar MRI imaging reveal that between half (47–66%) and just 2-10% of the variance might be attributed to physical loading and resistance training [44–46].

A diagnostic approach

Beginning with an accurate history and comprehensive physical examination with the necessary provocative testing, the initial evaluation of patients with low back pain begins. The subjectivity of patient reports of persistent spinal pain and the intrinsic challenge of isolating the target anatomic location during provocative testing without the impact of surrounding tissues confound these initial stages. Radiographic investigations, including plain film, CT, CT myelogram, and MRI, can help pinpoint a degenerative lesion or an area of nerve compression and can validate exam findings [25]. Additionally, there is frequently no correlation between the degree of anatomical or radiological abnormalities and the intensity of the symptoms [18]. The prevalence of degenerative alterations in asymptomatic patients accounts for the challenge in determining the clinical importance of detected radiographic changes in patients with LBP, even though there are associations between the quantity and severity of osteophytes and back pain [20, 22]. Electromyographic investigations showing normal distal motor and sensory nerve conduction studies with abnormal needle exam may also validate nerve compression complaints by clinical history. By isolating and anaesthetizing irritated nerve roots (through epidural) or by obstructing probable pain

generators within facet joints, sacroiliac joints, or the disc space itself (by discography), diagnostic injections can help with localisation [48].

Treatment options

Pharmacotherapy

Medication is frequently needed in addition to nonpharmacologic treatments to help manage pain and swelling, reduce disability, and enhance quality of life in patients with lumbar spondylosis. Numerous studies have examined the effectiveness of various oral drugs in the treatment of low back pain brought on by degenerative processes. Regarding the gold-standard strategy for pharmacologic therapy, there is still no definite agreement [49].

NSAIDS Since they provide analgesic and anti-inflammatory effects, NSAIDS are frequently viewed as a good first step in therapy. There is enough evidence to support its effectiveness in treating chronic low back pain [50–63], with GI issues being the most prevalent reason for use restrictions. In the long-term situation, COX2 inhibitors increase function while providing very limited alleviation from persistent LBP. Although they cause less GI problems, their usage has been restricted since there is evidence that long-term use increases cardiovascular risk [49].

Opioid medications

Opioid medications may be considered as an alternative or augmentive therapy for patients suffering from gastrointestinal effects or poor pain control on NSAID management. The practice of prescribing narcotics for chronic low back pain sufferers is extremely variable within practitioners, with Curr Rev Musculoskelet Med (2009) 2:94–104 99 a range of 3–66% of chronic LBP patients taking some form of opioid in various literature studies [54]. These patients tend to report greater distress/suffering and higher functional disability scores [55, 56].

Antidepressants

Given their claimed analgesic benefit at low doses, and dual role in treating commonly comorbid depression that accompanies LBP and may negatively effect both sleep and pain tolerance [52], antidepressants have also been extensively studied for the treatment of LBP symptoms. Antidepressants have been shown to relieve pain, according to two independent evaluations of the literature, but they have had no appreciable effects on functioning [57, 58].

Muscle relaxants

Antispasmodic or antispasticity drugs, which are muscle relaxants, may be helpful for chronic low back pain caused by degenerative diseases. Muscle relaxants are beneficial in terms of short-term pain alleviation and general functioning, according to a variety of research comparing benzodiazepines or nonbenzodiazepines with placebo [49, 59].

Injection therapy

A typical interventional technique for treating persistent axial and radicular pain brought on by lumbar spine degeneration is epidural steroid injections (ESI). These injections can be carried out using caudal, transforaminal, or interlaminar techniques. Local anaesthetic, steroid, contrast, and usually needles guided by fluoroscopy are injected into the epidural space at the target vertebral level and bathe departing nerve roots. There is a theory that suggests complementary pathways result in symptomatic alleviation. Local anaesthetics offer immediate confirmation of the diagnosis and, when used therapeutically, may interrupt the "pain spasm cycle" and stop the transmission of pain signals [60]. The ability of corticosteroids to decrease inflammation by blocking pro-inflammatory mediators is well known.

The number of ESI procedures carried out grew by 121% in the period of less than ten years (1998-2005) [60]. Despite their extensive use, there is ongoing debate about the effectiveness of these injections, which is stoked by their cost as well as the rare but possible hazards associated with the insertion of the needle and unfavourable drug reactions. Wide variations in reported success rates are cited in published data due to different study designs, unique procedural procedures, short cohorts, and insufficient control groups [61]. For instance, very few efficacy trials of lumbar ESI used fluoroscopy to determine the proper needle position before the year 2000. Even with competent clinicians, according to research, needle location may be improper up to 25% of the time without fluoroscopic guided confirmation [62]. To form conclusions on the effectiveness and value of ESI for the treatment of LBP, review articles and practising doctors must understand such methodological variations between research. One such review that looked at the effectiveness of interlaminar lumbar injections came to the conclusion that there was strong evidence for short-term pain relief and weak evidence for long-term benefit [60]. It cited, among many others, randomised controlled trials (RCT) by Arden and Carette of unilateral sciatic pain, which found statistically significant improvement in up to 75% of patients with steroid/anesthesia versus saline injections at 3 weeks, with benefit waning at 6 weeks and

Surgery is often saved for patients who have exhausted all other non-surgical treatments. In order to determine which patients are suitable "surgical candidates," factors including age, socioeconomic background, and anticipated degree of activity after surgery must also be taken into account [18]. To accomplish one of the two main objectives—spinal fusion or spine decompression—many surgical techniques have been devised (or both). Patients with spinal misalignment or excessive motion, such as with DDD and spondylolisthesis, are candidates for spinal fusion. There are various surgical fusion techniques, all of which involve adding a bone graft to develop between spinal components and reduce related mobility. Patients who clearly exhibit neural impingement should consider decompression surgery to treat the intrusion of bone or disc, which may be present in conditions such as osteophytosis, degenerative spondylolisthesis, disc herniation, or spinal or foraminal stenosis. There is still debate on the effectiveness of these procedures in treating chronic low back pain that is refractory to conservative therapy, despite the huge rise in surgeries over the past few decades. [72,73]

Conclusion

The diagnosis of lumbar spondylosis is challenging. Although there are many different definitions in the literature, we choose to define it generally as degenerative disorders of the spine. The correct diagnosis of symptomatic instances is highly challenging due to its widespread presence across all patient demographics, despite the fact that it may not be difficult to recognise radiographically. Despite significant research efforts to develop conservative and more invasive means of managing symptoms and delaying progressive decline, there is currently no definitive, gold-standard therapy approach to the varied spectrum of patient presentations. This field will continue to be a crucial topic of research given the prevalence of low back pain in the population and its social and economic ramifications. Risk factor research, genetic investigations, and experimental therapy methods have all provided significant hints. Efforts like these and others to follow will undoubtedly improve methods currently in use to address not only illness symptoms but also its development and, ultimately, prevention.

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